ANALYSIS OF 100 CASES OF PERFORATIVE PERITONITIS – STUDYING THE PROGNOSTIC FACTORS

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CERTIFICATE

This is to certify that this dissertation titled **"*ANALYSIS OF 100 CASES OF PERFORATIVE PERITONITIS – STUDYING THE PROGNOSTIC FACTORS*"** submitted by **DR.LUQMAN AHAMMED.P** to the faculty of General Surgery, The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of MS degree Branch I General Surgery, is a bonafide research work carried out by him under our direct supervision and guidance from October 2008 to October 2010.

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DECLARATION

I**, DR.LUQMAN AHAMMED.P** solemnly declare that the dissertation titled **"*ANALYSIS OF 100 CASES OF PERFORATIVE PERITONITIS – STUDYING THE PROGNOSTIC FACTORS*"** has been prepared by me. This is submitted to **The Tamilnadu Dr. M.G.R. Medical University, Chennai,** in partial fulfillment of the regulations for the award of MS degree (Branch I) General Surgery.

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CONTENTS

INTRODUCTION

Perforative peritonitis is a common emergency encounnted by surgeons all over the world. It needs prompt diagnosis and equally rapid measures to correct the underlying derangement.

The etiology of perforative peritonitis is varied and diverse, varying with the geographical location, culture characteristics and age of the patient among other factors. The spectrum of etiology of perforative peritonitis in tropical countries like India differ from its Western counter part, where lower Gastro intestinal perforation is common.

Duodenal ulcer perforation is the commonest type among Non-traumatic gastro-intestinal perforation. While the mortality of duodenal ulcer perforation has steadily declined all over the world in the last few decades it still remains a significantly mortal disease especially in developing countries.

While with the advent of better antibiotics enteric fever is easily curable, its complications like enteric perforation do occur from time to time primarily because of the failure to recongise therapy.

This clinical study is undertaken to evaluate the prognosis of various perforative peritonitis. Though the prognosis depends on multiple factors. I have taken some of the important prognostic factors which may probably affect the outcome of the disease.

HISTORICAL REVIEW

Rawlinson (1727) is credited with first published report of perforated gastric ulcer.

Hambergeri (1746) reported the first perforated duodenal ulcer.

Lombard (1989) is credited with the first laparoscopic repair of perforated duodenal ulcer.

A.K.C.LI (19992) reported the first sutureless laparoscopic repair of perforated duodenal ulcer.

Bright and Addison (1839) were the first to publish a report about perforated appendix.

SURGICAL ANATOMY

Peritoneum: The peritoneum is the serous membrane that forms the lining of the abdominal cavity .It is composed of a layer of mesothelium supported by a thin layer of connective tissue. The peritoneum both supports the abdominal organs and serves as a conduit for their blood and lymph vessels and nerves.

Although they ultimately form one continuous sheet, two types or layers of peritoneum and a potential space between them are referenced:

- The outer layer, called the parietal peritoneum, is attached to the abdominal wall.
- The inner layer, the visceral peritoneum, surrounding the viscera that are located inside the intraperitoneal cavity.
- The potential space between these two layers is the peritoneal cavity; it is filled with a small amount (about 50 ml) of slippery serous fluid that allows the two layers to slide freely over each other.
- The term mesentery is often used to refer to a double layer of visceral peritoneum. There are often blood vessels, nerves, and

other structures between these layers. The space between these two layers is technically outside of the peritoneal sac, and thus not in the peritoneal cavity.

Subdivisions

- There are two main regions of the peritoneum, connected by the epiploic foramen:
- The greater sac (or *general cavity of the abdomen*
- The lesser sac (or *omental bursa*.‘ The lesser sac is divided into
- The lesser omentum (or *gastrohepatic*) is attached to the lesser curvature of the stomach and the liver.
- The greater omentum (or *gastrocolic*) hangs from the greater curve of the stomach and loops down in front of the intestines before curving back upwards to attach to the transverse colon.

The mesentery is the part of the peritoneum through which most abdominal organs are attached to the abdominal wall and supplied with blood and lymph vessels and nerves.

Functions of peritoneum:

- Pain perception
- Visceral lubrication
- Fluid absorption
- Inflammatory and immune response
- Fibrinolytic activity

STOMACH:

Stomach is the hollow viscus connecting esophagus and duodenum. It secretes Hydrocholoric acid. Due to reservoir function it has a variable shape.

PARTS OF STOMACH:

1. Fundus 2. Body 3. Antrum 4. Pylorus.

Pyloric sphincter consists of inner circular and outer longitudinal muscle which form a functional entity contracting concentrically to constitute the emptying mechanism of stomach.

BLOOD SUPPLY

1. Lesf gastric vessels
2. Right gastric vessels

3. Short gastric vessels
4. Right and left gastric – epiploic vessels.

All are branches from celiac axis.

MICRO CIRCULATION

There is a rich sub-mucous plexus except at the lesser curvature, where the blood supply comes from direct branches of right and left gastric arteries. Due to long course of these vessels through serosa to mucosa, ischaemia and ulceration are common in lesser curvature.

DUODENUM

Duodenum is 25cm long. Makes a 'C' shaped bend and embraces the head of pancreas. Has four parts. First part: 5cm long, continues from pylorus. Extends upwards and backwards to the right of upper border of L1 vertebra. Second Part : 7.5cm long, extends down to L3 vertebra. Third & Fourth Part : Extends to the left.

PERITONEAL COVERING:

First Part : The convexity and anterior surface of "C" is covered with peritoneum.

Other parts of duodenum are devoid of peritoneum. Duodenal ulcer commonly occurs in the first part. The anteriorly placed ulcer

perforates. The posteriorly placed ulcer penetrates and causes massive bleeding due to erosion of gastro duodenal artery.

SMALL INTESTINE

The average length is seven meters. The upper two fifth is the jejunum and the lower three fifth is the ileum.

It is covered entirely by peritoneum.

The mesentery extends from the left of seconds lumbar vertebra to the right sacro-iliac joint and it contains superior mesenteric artery and tits branches.

The jejunum has one or two arterial arcades and the vasa brevia are long.

The ileum has two or three arterial arcades and the vasa brevia are short.

There is a rich network of sub mucosal lymphatics.

The terminal ileum has abundant lymphoid tissue aggregations called Peyer's patches.

APPENDIX

It is approximately 9cm long. It lies near the ilecaecal junction. Its wall contains lymphoid tissue. It is supplied by appendicular branch of ileo-colic artery. The location of the base of appendix depends upon the position of caecum. It may be subhepatic or lie in the right iliac fossa.

Tip of appendix can occupy different positions and is usually retrocaecal. It may also occupy pre-ileal, pelvic, paracolic positions.

LARGE INTESTINE

Extends from ileum to anus. It is approximately 135 cm long.

Circular muscle layer is continuous. Outer longitudinal muscle layer is arranged in three bands – Taenia coli.

Transverse and sigmoid colon have a long mesentery and is covered by peritoneum all around.

Caecum has peritoneal covering.

Rectum : 12cm long. Starts at S-3 level and terminates 3cm in front and below tip of coccyx.

Peritoneal covering (rectum)

Upper one – third –is covered anteriorly and laterally.

Middle one – third – anteriorly.

Lower one – third – (not covered) Extra peritoneal.

BLOOD SUPPLY

Middle colic, right colic, ileocolic arteries

Leftcolic, sigmoidal and rectal arteries

Marginal artery is the paracolic vessel of anastamosis between colic areteris.

GALL BLADDER

A pear shaped organ. 7.5cm – 12.5cm long. Its normal capacity is 50ml

PARTS

1. Fundus. 2. Body 3. Neck

Muscle fibers arranged in a criss cross manner

Crypts of Luschka is present in the mucous membrane.

Cystic duct is 2.5cm long it contains spiral valve of Heister.

Blood Supply: Cystic artery is a branch of right hepatic artery.

FUNCTIONS

1. Reservoir of bile
2. Concentration of Bile
3. Secretion of mucus

REVIEW OF LITERATURE

INTRAABDOMINAL INFECTIONS :

Microbial contamination of the peritoneal cavity is termed peritonitis or intraabdominal infection, and is classified according to etiology.

Primary microbial peritonitis occurs when microbes invade the normally sterile confines of the peritoneal cavity via hematogenous dissemination from a distant source of infection or direct inoculation. This process is more common among patients who retain large amounts of ascites, and in those being treated for renal failure via peritoneal dialysis. These infections invariably are monomicrobial and rarely require surgical intervention. Treatment consists of administration of an antibiotic to which the organism is sensitive. Removal of indwelling devices (e.g., peritoneal dialysis catheter or peritoneovenous shunt) may be required for effective therapy of recurrent infections.

Secondary microbial peritonitis occurs subsequent to contamination of the peritoneal cavity because of perforation or severe inflammation and infection of an intraabdominal organ. Examples

include appendicitis, perforation of any portion of the GI tract, or diverticulitis. Effective therapy requires source control to resect the diseased organ; debridement of necrotic, infected tissue and debris; and administration of antimicrobial agents directed against aerobes and anaerobes. Effective source control and antibiotic therapy is associated with lowfailure rates and a mortality rate of approximately 5–6 percent; inability to control the source of infection leads to mortality greater than 40 percent. The response rate to effective source control and use of appropriate antibiotics has remained approximately 70–90 percent over the past several decades.

Patients in whom standard therapy fails develop an intraabdominal abscess, leakage from a gastrointestinal anastomosis leading to postoperative peritonitis, or tertiary (persistent) peritonitis. The latter is a poorly understood entity that is more common in immunosuppressed patients in whom peritoneal host defenses do not effectively clear or sequester the initial secondary microbial peritoneal infection. Unfortunately, even with effective antimicrobial agent therapy, this disease process is associated with mortality rates in excess of 50 percent.

PATHOPHYSIOLOGY OF PEPTIC ULCER

Gastric secretion aids in breakdown of food into smaller particles. About 2 litres of gastric secretion is produced every day.

PHASES OF GASTRIC SECRETION

Cephalic phase – Initiated by thought, sight, smell and taste.

Gastric phase – entry of food in stomach initiates it.

Intestinal phase – When chyme begins to empty into duodenum this phase in initiated.

GASTRIC SECRETARY CELLS

1. Oxyntic glands – Oxyntic cells secrete HCL & Intrinsic factor

Chief cells secrete Pepsiongen. Mucus cells secretes muscus

2. Pyloric glands – G cells secrete Gastrin.

Hydrochloric acid is a major etiological factor in acid peptic diseases.

Acetyl choline, histamine and gastrin – stimulate HCI secretion.

Somatostatin inhibits HCL secretion.

GASTIRC MUCOSAL BARRIER

This protects gastric lining cells from auto digestion by HCL. Turnover of gastic mucus cells is 5 x 105 cell . minute.

MUCOSAL DEFENSE MECHANISMS

Surface epithelial cells secrete mucus and bicarbonate, lowering the acid environment to neutral mucous surface.

Gastric mucosal cells have a specialized apical surface membrane that resist diffusion of acid back into the cells.

Prostaglandins enhance resistance of mucosa to injury by maintaining mucosal blood flow and stimulating secretion of mucus and bicarbonate.

MECHANISM OF ULCER FORMATION

Break – down of mucosal defence mechanism.

Increased acid pepsin secretion.

Chronic ulcers – solitary in 80% of cases.

98% are located in proximal duodenum or antrum of stomach.

Macroscopy : Punched out. Ulcer 2-4cm diameter penetrates into tunica muscularis. Ulcer base in clean.

Microscopy : Varies with activity and degree of ulcer healing.

Active lesions have :

- Surface Fibrin.
- Inflammatory layer
- Zone of granulation tissue.
- Fibrous scar with blood vessel.

GASTRIC AND DUODINAL ULCER PERFORATION

Perforation of peptic ulcer is one of the most dramatic complication of peptic ulcer and inspite of modern management is still a life threatening catastrtophe. Perforation is an unmistakable event and it provides a useful marker in epidemiologic event and it provides a useful marker in epidemiologic studies of peptic ulcer disease. Difficulties in studying natural history of perforated peptic ulcer are compounded by the fact that ulcer patients are notoriously difficult to follow up because they tend to belong to lower socio-economic group and often have an unstable life style.

Perforation of peptic ulcer is more common in men than women. M:F = 2:1

Incidence is high in middle age patients

Incidence of perforated duodenal ulcer to perforated gastric ulcer is 25:1,8:1

Most frequent in semiskilled and unskilled workers.

Independent of smoking habits.

No worldwide constistency in seasonal patterns.

AETIOLOGY OF PERFORATION PEPTIC ULCER

Precipitated by large meal, exertion, trauma.

Drinking chilled beer liberates carbondioxide causing rise in intra gastric pH

ASSOCIATION

Burns, Neurological injury.

Zollinger Ellison syndrome

Aortic aneurysm surgery

NSAID INGESTION

- Due to systemic inhibition of prostaglandin Production
- Safe ranges of NSAID are not indetifiable.
- Not dependent on duration of usage.
- Effectively prevented by cotherapy with prostaglandin analogues.

CIGARETTE SMOKING

- Impairs ulcer healing
- Promotes recurrence
- Increases surgical risks.

HELICOBACTER PYLORI

- H.Pylori is a small curved gram negative micro serophillic rod with multiple polar flagellae. Stomach is its normal habitat. It lies closely opposed to gastric mucus secreting cells. Ammonia generated by H.Pylori alters gastric epithelial permeability, impairs ionic integrity of mucous resulting in mucosal injury. H.pylori has a role in non-ulcer dyspepsia, gastritis. No

direct relationship between H.pylori and duodenal ulcer disease has been proved. But eracication of H.pylori reduces ulcer recurrence rate. The role of H.pylori in perforated peptic ulcer diseases is not clear. The H.pylori positive rate among perforated duodenal ulcer disease ranges from 28% to 48%. All patients with H.pylori infection should be treated with eradication therapy as it speeds up healing and decreases the rate of ulcer disease.

PATHOLOGY

1. Acute perforation.
2. Subacute perforation.
3. Chronic perforation.

Acute perforation is by far the commonest presentation.

Dent et al (1977) found that, biopsy specimen of perforated ulcer edge had an element of fibrosis. So it is usually the chronic ulcer which perforates. It is reasonable to speculate that perforation is due to sudden sloughing of an unsuspected portion of the floor of ulcer because of impairment of blood supply by end – arteritis and the

ragged hole is rapidly digested by flow of gastric contents until dense fibrous tissue around there fim of ulcer is exposed.

SITE

Kozoll meyer etal (1962) found that

- Anterior wall of first part of duodenum perforates in 92% of cases; in 2% of cases – posterior wall perforates and in 6% of cases – pyloro duodenal junction perforates.

Posterior ulcer usually penetrates. But may perforate extra peritoneally and collect in perinephric region. It may track down through paracolic gutter and may end as duodenal fistula on incising the abscess. Size of duodenal ulcer perforation varies from 2mm to 10mm.

BACTERIOLOGY

Frequency of positive cultures increases with time and is high after 48 hours.

Organisms found usually are Coliform, streptococci, anaerobic organisms.

ACUTE PERFORATION

STAGES

1. Stage of peritoneal irritation.
2. Stage of peritoneal reaction.
3. Stage of bacterial peritonitis.

PRIMARY STAGE

STAGE OF PERITONEAL IRRITATION

Due to escape of gastric and duodenal contents.

Lasts for 2-6 hours.

SIGNS

Patients avoid movement.

Respiratory rate is increased. Pules rate is – normal

Board like rigidity of abdomen is present.

Presence of rebound tenderness.

Total absence of bowel sounds.

Back pain – if it perforates into lesser sac.

SECONDARY STAGE

(STAGE OF PERITONEAL REACTION)

Occurs after 2 hours – 6 hours. Lasts 6 hours

Tachypnoea , tachycardia,

Liver dullness is obliterated.

Distension increases as a result of paralytic ileus.

Septicaemia and multi-system failure frequently supervene.

INVESTIGATION

Plain X-ray abdomen erect including diaphragm after nasogastric aspiration. Pneumo peritoneum is see in 80% of cases. Even one c.c of free gas can be detected in left lateral decubitus film.

SUBACUTE PERFORATION

An ulcer may perforate and seal rapidly before there is spillage of gastric and duodenal contents into peritoneal cavity.

Sudden onset of acute upper abdominal pain

Local tenderness and rigidity (Upper abdomen)

D.D. Acute cholecystitis.

INVESTIGATION

Ultrasound abdomen – to rule out gallbnladder pathology.

CHRONIC PERFORATION

When an ulcer perforates into an area that is walled off by adhesions (or) by adjacent viscera like colon, greater omentum (or) when a gastric ulcer preforates into an omental sac with sealing off of the omental foramen – chronic abscess may form.

- No features of peritonitis.
- X-ray abdomen - may reveal a subphrenic cavity containing gas.
- USG / CT scan – may be useful.
- OGD may allow identification of peptic ulcer as a cause of abscess cavity.

MANAGEMENT

Aims are

Avoidance of mortality

Permanent cure of ulcer and its complications

Avoidance of post operative sequelae.

RISK FACTORS:

- Boey et al (1982) have identified three main risk factors.
- Concurrent medical illness – like cardio respiratory disease, renal failure, diabetes mellitus, hepatic precoma.
- Pre operative shock.
- Perforation more than 24 hours.

Old age, ulcer history and extent of peritoneal soiling were not found to be significant risk factors.

RESUSCITATION:

Adequate resuscitation with crystalloids and colloids is important.

MANAGEMENT:

A. Conservative

B. Surgical intervention

- Simple closure of perforation with a live omental patch.
- Simple closure with a free omental patch.
- Simple closure wht definite ulcer operation.

- Laproscopic suture repair of perforation.
- Laproscopid fibrin glue repair.

CONSERVATIVE TREATMENT

Described 50 years ago. Aim is to allow location and spontaneous sealing of ulcer to occur. This policy is rarely used now because of safe modern anaesthesia and resuscitation. It has a place in two groups of patients.

1. Elderly patient who present late with localized / sealed perforation.
2. In the severely ill patient with all three risk factores (Boey et al 1987). At present even modern surgery is unable to prevent 100 percent mortality in this small group of patients.

Objections to Non-Operative Treatment

1. Uncertainty or error in diagnosis.
2. Site of perforation is unknown.
    a. Perforated gastric ulcer has a higher death rate than perforated duodenal ulcer if conservative management is followed.

SURGICAL LINE OF MANAGEMENT

GRAHAMS PATCH

Here simple closure with a free omental patch is used using absorbable suture material.

When perforation is large extended pyloroplasty (Passos et al 1986) or various forms of gastric resection will be useful. Other procedures which are tried are duodenal intubation, Staplers.

Mortality of simple closure varies from 0-16.6%

Complications of simple closure

1. Gastric outlet obstruction 26% (Play worth et al)

SIMPLE CLOSURE WITH DEFINITIVE ULCER OPERATION

Definite indications are:

- Presence of synchronous second ulcer complications.
- Previous ulcer complications.
- Perforation of ulcer during anti secretary treatment.

Relative indications:

- Long preperforation ulcer history.
- Young patient.

Immediate definite operation is contra – indicated, if the patient is a poor risk because of major concurrent medical illness or shock or more than 24 hours old perforation.

DEFINATE PROCEDURES ARE

Proximal gastric vagotomy

Truncal vagotomy with posterior gastro – jejunostomy.

Antrectomy.

Martality of PVG – 4.5% (Jordan Etal)

TV & drainage 0% (Boey et al)

Age by itself was not a factor.

LAPAROSCOPIC SUTURE REPAIR

Using laproscope suturing is carried out by passing a needle through the duodenum near the perforation and through a mobilized patch omentum. An extra corporeal roeder knot is tied in the suture and passed down to fix the patch over perforation. This is followed by peritoneal lavage with normal saline.

LAPAROSCOPIC FIBRIN GLUE REPAIR

A piece of gelatin sponge (spongostan0 20x15x10mm thick sheet is rolled into a cone. This plug is place into the perforation so that base of the cone protrudes into serosal surface. A pre warmed two milliliter volume of two component fibrin sealant is injected slowly through a double lumen catheter around the plug to secure it. peritoneal lavage is done with normal saline.

SUMMARY

The basis of treatment for duodenal ulcer perforation is surgical intervention with oversewing in majority of patients.

Definitive surgical treatment is strongly advocated for patients with no risk and who have associated complications like bleeding and obstruction.

Non operative measures are suggested for patients with major risk, or localized peritonitis.

Although definitive surgery in good hands is safe the price of unnecessary surgery and permanent side effects should not be ignored.

REPAIR OF THE DIFFICULT DUDENAL PERFORATION

A difficult duodenal perforation can be define as a duodenal perforation which is either difficult to diagnose or difficult to repair. Various modalities of treatment advocated for such a situation are:

1. Closure of the perforation by oemntal implantation.
2. Closure of the perforation by an omental patch.
3. Closure of the perforation using falciform ligament
4. Jejuna serosal patch technique
5. Roux-en-Y duodenojejunostomy
6. Pyloroplasty
7. Operations involving exclusion or diverticulization, including partical gastrectomy or gastric dissociation.
8. Duodenostomy
9. Resection.

CLOSURE OF THE PERFORATION BY OMENTAL IMPLANTATION

Perforation is repaired by drawing and implanting a protion of omentum in to the perforated site. Omentum gets firmly adherent and under goes inflammation necrotic changes granulation, and reduction in size and fibrosis. There is no luminal obstruction. The defect decreases rapidly in its size and healing complete with in eight weeks. This procedure is simple, dependable and does not require expertise. Indication is seriously sick patient with a giant peptic perforation, edges of which are quit friable either as a primarly procedure or if the sutures placed a the first operation have given way.

FREE OMENTAL PLUG:

A free omental graft of suitable dimensions is cut, rolled and fashioned into the shape of mushroom and fixed to the perforated site. Some people feel that the omental plug can be rendered ischemic by tying 3 sutures across it to hold it. over the perforation such ischemia may give rise to necrosis with subsequent leakage. They advocate pulling in of the omental plug by attaching it with a cargut tie with to the nasogastric tube which is manipulated by the surgeon and made to protrude out of the perforation. The edges of omental plug are further

tucked to the intestinal wall. The nasosgastic tube is placed on a low pressure suction for 72 hours and removed after seven days, by which time the catgut suture attaching it to the omentum has dissolved away.

PERFORATED GASTRIC ULCER

Gastric ulcer perforation has a frequency of one fifth to one – tenth of that of duodenal ulcer, but carries a five fold increases in mortality about 25-30% (Stein – Heber 1985).

Elderly females with dual complications of bleeding and perforation are at a high risk. Smoking and NSAIDS increase the risk (Wilson et al 1985).

Many patients have no dyspeptic history.

Usually patients present late leading to localized perigastric abscess. Upto 10% perforated gastric lucers may be malignant.

MANAGEMENT

Majority are situated in the antrum or along lesser curvature of stomach. Ideally optimal procedure is gastric resection and a Billroth I gastroduodenal anastomosis. However many patients are ill and so

1. Four quadrant full thickness blopsy and oversewing.

2. Truncal vagotomy and pyloroplasty are alternatives.

The use of potent H2 Receptor antagonist may possibly reduce the higher recurrence and reperforation chance associated with simple overswe (Wilson-Macdonald et al 1985)

- Emergency gastrectomy carries a mortality of 43% varus 32% simple oversaw (Collier and Pain 1985)

- Perforation of gastric carcinoma is fortunately rare 2% incidence rate. It is a bad prognostic factor.

- Resection should be considered in most patients but is not feasible in all patients.

- Chemo sensitive tumor s- Non Hodgkins lymkphoma may perforate during chemotherapy.

TYPHOID PERFORATION

A distinctive acute systemic febrile infection caused by salmonella Typhi, salmonella paratyphoid A & B. They are nonencapsulated gram negative bacilli.

Incubation Period 3-60 Days

Poor sanitation and poor hygiene explains its common occurrence. Peyer's patches in terminal ileum show hyperplasia, necrosis and ulceration. Ulcer heal without scarring. Ulcer are oval and placed longitudinally. Spontaneous separation of slough from ulcer leads to hemorrhage and perforation. Perforation may also be due to vascular thrombus in Peyer's patches, and steroid therapy.

Incidence : 1% - 3% Of Typhoid Fever

Symptoms complex of fever, headache, macular rash in upper abdomen, splenomegaly, vomiting, abdominal pain and distension with tenderness and guarding are highly suggesting of typhoid perforation.

Plain X – ray abdomen – shows multiple fluid levels.

Air under diagram may be present

WBC count – raised.

Blood culture –will be positive for enteric group of organisms in 60% of cases.

Widal though useful is non specific; is positive in 25-75 % of cases.

Bone marrow, culture – is positive in 90% of cases.

MANAGEMENT

NON – OPERATIVE MANAGEMENT

Huckstep advocated conservative treatment (ie) Antibiotic and Ochsner Sherren Regime for typhoid perforation because lower ileum is paper thin and is liable to perforate at more than one spot and repair in a friable gut an almost impossible task. The ileum is linked to a wet blotting paper.

SURGICAL MANAGEMENT:

Frankilin favoured surgical approach, as closure of perforation eliminates continued contamination and lessens toxaemia. Surgical treatment is essential because peritonitis is poorly localized and omentum does not seal off the perforation.

Mortality of operative treatment is one of the decline because of better antibiotics, good anaesthesia, improved surgical technique and better post operative care.

Mortality rises as perforation – operation time interval increase. It is 14% at 24 hours, 22.8% at 48 hours 31.3% at 72 hours.

If perforation is more than tendays old critical period of toxaemia is over and so at times it is safer to persist with conservative treatment and only drain residual abscesses.

SURGICAL PROCEDURES

Drainage alone

Simple closure in two layers

Simple closure with exteriorization of sutures loop.

Wedge excision of ulcer and closure of ileal defect.

Simple closure of perforation with ileo-transverse anastmosis.

Resection of most affected loop of ileum an anastmosis in two layers.

Deliberate ileal fistula

Drainage alone : Has very high mortality.

SIMPLE CLOSURE IN TWO LAYERS

After freshening the edges simple closure in two layers is the commonest procedure performed. Non absorbable suture material is advisable. There are more chances of breakdown and high fistula rate.

Mortality.31%

SIMPLE CLOSURE WITH EXTERIORIZATION OF SUTURED LOOP

After closure of perforation the entire loop of bowel including suture line is brought out at the lower end of incision. The peritoneal edge and muscle are sutured to bowel wall with non-absorbable sutures.

Nadkarni et al reported faecal fistula in 10 out of 16 cases, which later closed spontaneously. Mortality rate was 12.5% when compared to 43.5% for non exteriorized group.

SIMPLE CLOSURE OF PERFORATION WITH ILEO TRANSVERSE ANASTMOSIS

Eggleton etal compared this with primary closure alone. There was no different between either group in mortality and morbidity but a reduction in major complications in by pass group from 74.4% to 47.6%.

RESECTION OF MOST AFFECTED LOOP OF ILEUM

Done if fragile, edematous or multiple or perforations are present. Resectionshould include all diseased part and a two layered anastomosis is done.

DELIBERATE ILEAL FISTULA

In critically ill patients an ilieostomy through site of perforation is a simple safe short procedure and may be life saving. Maloney had 100% success with this procedure in five patients. Chambers had a 25% mortality among 12 patients.

COMPLICATIONS OF ENTERIC PERFORATION

Toxemia	Incisional Hernia
Hypotension	Reperforation
Wound Dehiscence	Intestinal Obstruction
Faecalfistula	Renal failure.

ACUTE APPENDICITIS AND PERFORATION

Acute appendicitis is still a common surgical emergency. Early diagnosis and prompt appendectomy is the goal. Appendectomy should be avoided in the presence of established mass. In infants and elderly, in case of real doubt, decision should be towards immediate appendectomy.

Age : peak incidence is in childhood

Males are affected more often

There is an unusual but accepted familial susceptibility.

PATHOLOGY

Two types

1. Non obstructive acute appendicitis
2. Obstructive acute appendicitis.

1. Non obstructive

Inflammation commences in mucous membrane and lymph follicles. It proceeds and terminates by resolution, ulceration suppuration, fibrosis, gangrene.

2. Obstructive Type

Two out of three cases of the obstructive type. Obstruction can be in the

a) Lumen- faecolith, foreign body or parasites
b) Wall- inflammatory, rarely malignancy
c) Out side – Kinking, adhesions

The products of inflammation are pent up. Often within 12-18 hours, appendix distal to obstruction becomes gangrenous. Perforation occurs

most often at the site of an impacted faecolith, Subphrenic and pelvic abscesses are common sequelae.

CLINICAL FEATURE

-abdominal pain which shifts from umlical region to the right iliac fossa

- Vomiting due to protective pylorospasm
- Localized tenderness – at right iliac fossa
- Rigidity in right iliac fossa
- Later generalized rigidity.

MANANGEMENT:

Appendectomy and peritoneal toileting

Drainage of peritoneal cavity is indicated only in rare instance

GALL BLADDER RERFORATION

The gallbladder may perforate in acute calculous cholecystitis. In 95 percent of cases gallstone is found impaced in Hartmann's pouch or obstructing the duct, Which cases perforation Bacteria can be cultured in most cases, common organisms are Ecoli, Kleibsiella and streptococcus faecalis.

Gas forming organisms may rarely infect the gall bladder.

PERFORATION OF GALL BLADDER

SITE

1. Usually at founds, Due to diminished blood supply.
2. Neck- due to pressure necrosis of an impacted calculus.

SEQUELAE

1. Local abscess – due to adhesions between gallbladder, and greater omentum.
2. Perforation into general peritoneal cavity-occurs in 0.5% of acute choloecystitis. Usually men are affected and mortality is about 50%.

CLINICAL FEATURES

- Sudden pain – right hypochondrium
- Pyrexia
- Severe nausea and vomiting
- Tenderness and rigidity

INVESTIGATION

- Plain X-ray abdomen
- Rarely gas in gall bladder may be seen
- Ultrasound abdomen
- Gall stones can be found

- Wall thickness can be assessed
- Localised abscess can be detected.

TREATMENT

Cholecystectomy (emergency)

COLONIC PERFORATION

THE COMMON CAUSES ARE

1. Amoebic colitis – perforation
2. Diverticultis - perforation
3. Colonic malignancy - perforation
4. Ulcerative colitis and chrons disease - perforation

ADENO CARCINOMA OF COLON

In colonic malignancy usually the annular type causes perforation due to proximal distension.

Direct infiltration may also cause perforation. Direct infiltration occurs usually in ulcerative type.

Perforative peritonitis in colonic malignancy is a bad prognostic factor.

SITE OF PERFORATION IN COLONIC MALIGNANCY

1. Local infiltration

2. Caecal blow out (if ileocaecal valve is patent)

PROGNOSIS

Usually carriers a bad prognosis because of faecal peritonitis and poor general condition of patient.

AIM OF STUDY

The aim of my study is to study the prognosis of various perforative peritonitis on the basis of

1. AGE OF THE PATIENT
2. SITE OF PERFORATION
3. SIZE OF PERFORATION
4. TIME OF PRESENTATION

MATERIAL AND METHODS

This is a prospective study has been based on the analysis of 100 cases of perforative peritonitis admitted under the department of General Surgery, Government Rajaji Hospital, Madurai from October 2008 to October 2011. Cases were admitted as emergency and possible immediate investigations were done.

Exclusion Criteria:

1. Traumatic perforations were excluded
2. Appendicular mass excluded.

Statistical Tools

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using **Epidemiological Information Package (EPI 2008)** developed by Centre for Disease Control, Atlanta.

Using this software range, frequencies, percentages, means, standard deviations, chi square and 'p' values were calculated. Kruskul Wallis chi-square test was used to test the significance of difference between quantitative variables. A 'p' value less than 0.05 is taken to denote significant relationship.

RESULTS AND DISCUSSION

Incidence Table: 1 Size of Perforation

Site of Perforation	Cases	
	No	**%**
A	15	15
DU	61	61
G	2	2
C	1	1
I	18	18
Unknown	3	3
Total	100	100

In my study of 100 cases of perforation peritonitis : the incidence of duodenal ulcer perforation is highest 61% followed by

Ileal perforation 18%

Appendicular Perforation 15%

Gastric Perforation 2%

Colonic Perforation 1%

PROFILE OF CASES STUDIES

Table : 1 Age Distribution

Age in Years	**Cases**	
	No	**%**
Less than 20 years	7	7
20-29	22	22
30-39	38	38
40-49	16	16
50-59	8	8
60 & Above	9	9
Total	100	100
Range	15-70 Years	
Mean	36.9 Years	
S.D	13.3 Years	

Age varied from15 yrs to 70 years. Youngest was 15 years with appendicular perforation, oldest was 70 years old with duodenal perforation.

Mean age is 39.9 years with SD 13.3

Table : 2 SEX DISTRIBUTION

Age in Years	Cases	
	No	**%**
Male	89	89
Female	11	11
Total	100	100

Sex ratio irrespective of pathological condition Male : Female is 8.09:1 which is comparable with other studies.

Table : 4 Time of presentation the onset (in days)

Time of presentation after onset (in days)	Cases	
	No	**%**
< 1 day	6	6
1 Day	28	28
2-3 Days	53	53
>3 days	13	13
Total	100	100

After onset of symptoms patients presented to us within 6hrs to 6 days.

Mean time of presentation is 2.14 days with SD 1.22 day.

Table 5: Size of Perforation

Size of Perforation	**Cases**	
	No	**%**
Up to 1 x 1	65	77.4
>1 x 1	19	22.6
Total	84	100

77.4% of cases had perforation size ≤1cm (maximum diameter), Remaining 22.6% had perforation size >1cm.

Table 6: Outcome

Out come	**Cases**	
	No	**%**
Recovered	84	84
Death	16	16
Total	100	100

Over all mortality in my study is 16% is favourably comparable in with other published series.

Table 7 : Age and Outcome

Outcome	**Age in Year (Mean ± SD)**
Recovered	**34.6 ± 12.1**
Dead	**48.9 ± 13.3**
P	**0.0004 Significant**

Age Group	Out come			
	No	%	No	%
<20 Years (7)	7	100	-	-
21-30 (22)	20	90.9	2	9.1
31-40 (38)	12	94.7	2	5.1
41-50 (16)	4	75	4	25
51-60 (8)	5	50	4	50
>60 (9)	84	55.6	16	44.4
Total (100)		84		16
P	0.0004 Significant			

Mean age of patients who died is 48.9 with SD 13.3 and mean age of patients who recovered is 34.6 with SD 12.1

P value is 0.0004 which is significant, indicating mortality increases with age.

Site of perforation and outcome

Site of perforation	Out come			
	Recovered		Death	
A (15)	15	100	-	-
DU (61)	55	90.2	6	9.8
G (2)	1	50	1	50
C (1)	-	-	1	100
I (18)	13	72.2	5	27.8
UK (3)	-	-	3	100

Overall mortality irrespective of site of perforation of 16%

3 patients were not fit for surgery due to hypotensive shock and bilateral flank drain was done, but all the three patient died.

One case was of colonic perforation, a 55years old male underwent laparotomy for peritonitis showed sigmoid colon growth with caccal blow out and feeal peritonitis. But patient died during surgery.

In the study colonic perforation and patients managed conservatively had maximum mortality (100%) followed by

gastric perforation	50%
Ileal perforal	27.8%
Deodenal Perforation	9.8%
Appendicular Perforation	0%

Site of perforation and complication

Site of perforation	Complications			
	Yes		No	
A (15)	7	46.7	8	53.3
DU (61)	28	45.9	33	54.1
G (2)	-	-	2	100
C (1)	-	-	1	100
I (18)	10	55.6	8	44.4
UK (3)	3	100	-	-

In this study morbidity is assessed based on occurrence of complications like sepsis, hypotension, burst, abdomen, respitary complication feeal fistula, brilary leak.

3 patients who were managed conservatively had hypotension.

Among those perforation, where site is known, ileal perforation got maximum incidence of complications 55.6 following

Appendicular Perforation	46.7%
Duodeanal	45.9%
Gastric	0%
Colonic	0%

Site of Perforation and Duration of Hospital stay

Site Perforation	Duration of Hospital stay (Mean ± SD)
A DU G C I UK	12.9 ± 6.1 13.1± 6.9 8.5 ± 2.5 1 6 ± 10.9 1.3 ± 0.6
'p'	0.0065 Significant

In this study mean hospital stay is maximum for Ileal perforation, i.e. 16 day with SD 10.9 days.

Followed by at least for gastric perforation is 8.5 day with SD 2.5 day.

P Value is 0.0065, which D. significant.

Size of Perforation and outcome

Size of perforation	Outcome			
	Recovered		Death	
Upto 1 x 1 (65) 1 x 1 (19)	57 14	87.7 73.7	8 5	12.3. 26.3
P Vale – 0.1317 (not significant)				

In my study size of perforation does not influence outcome

Associated factors and outcome

Associated Factors	Outcome			
	Recovered		Death	
Present (40) Absent (60)	35 49	87.5 81.7	5 11	12.5 18.3
P Value o.6163, which is not significant				

Associated factors the smoking, alcoholism, NSAID, APD etc do not found to have any effect on out come.

Comorbid conditions and outcome

Comobid conditions	Outcome			
	Recovered		Death	
Present (11)	4	36.4	8	63.6
Absent (89)	80	89.9	7	10.1
P Value 0.0002 which is statistically significant				

Comorbid condition increases mortality.

Time of presentation and outcome

Time of presentation after onset	Outcome			
	Recovered		Death	
<1 day (6)	6	100	-	-
1 day (28)	28	100	-	-
2-3 days (53)	46	86.8	7	13.2
>3 days (13)	4	30.8	9	69.2
Average time (in days)	1.83 ± 0.95		3.75 ± 1.24	
P Value is 0.0001, which is Significant				

All patients who presented in ≤ 24hrs recovered (100%). As time of presentation increases mortality increases.

DISCUSSION

Perforation peritonitis is a frequently encountered surgical emergency in tropical countries like India, most commonly affecting young men in the prime of life as compared to the studies in the west where the mean age is between 45–60 years. In majority of cases the presentation to the hospital is late with well established generalized peritonitis with purulent/fecal contamination and varying degree of septicemia. The signs and symptoms are typical and it is possible to make a clinical diagnosis of peritonitis in all patients.

The perforations of proximal gastrointestinal tract were six times as common as perforations of distal gastrointestinal tract as has been noted in earlier studies from India which is in sharp contrast to studies from developed countries like United States, which revealed that distal gastrointestinal tract perforations were more common.

There were 16% deaths which is comparable with other published series. Peritoneal contamination is a crucial consideration in patients with peritonitis and problem of mortality is a problem of infection. So by early surgical intervention, we succeed in preventing further contamination by removing the source of infection though the

end result will also depend upon the general host resistance and the antibiotic sensitivity of the organism.

The major cause of postoperative morbidity were wound infectiond, septicemia and respiratory complications e.g. pneumonia, atelectasis, pleural effusion or ARDS, which are preventable and should be detected early and aggressively treated.

In this study mortality is significantly associated with age of patients, time of presentation, site of perforationadn associated comorbid factors like DM, HT, COPD etc.

Boey et al (1982) have identified three main risk factors as

- Concurrent medical illness – like cardio respiratory disease, renal failure, diabetes mellitus, hepatic precoma.
- Pre operative shock.
- Perforation more than 24 hours.

Most of the patients presented late with mean time of presentation is 2.14 days. This because of lack of awareness and delay in diagnosis, delay in diagnosis is more commonly seen in ileal perforamtion because patient were being treated for typhoid fever and inattention to the peritoneal signs which developed early in due course

of the disease. So, the contamination is more, which leads to post operative mortality due to septicemia, and morbidity like wound sepsis. Delayed wound healing may also be associated with poor nutrition.

Though some studies show size of perforation affect the prognosis, in this study there was no significant association between size of perforation and over all outcome.

In this study, no mortality found in appendicular perforation, but wound infection was commonest complication due to appendicular perforation.

Since number of cases in colonic and gastric perforation is small, long term studies are needed to assess the complication rate and mortality due to them.

Average hospital stay for ileal perforation (16 days) is > duodenal perforation (13.1 days) > apendicular perforation (12.9 days) indicating the postoperative morbidity in the same order.

Patients with comorbid condition like DM, HT, ARF, COPD etc found to have poor outcome.

In short, outcome of perforative peritoritis is depends on multiple factors, some are known, other are unknown, which is to be kept in mind while treating patients with perforative peritoritis and patients should be managed accordingly.

CONCLUSION

1. In non-Traumatic gastro – intestinal perforation duodenal ulcer perforation is the commonest (61)
2. Males are more commonly affected than females (8.09:1)
3. The incidence on Non-traumatic gastro – intestinal perforation is high in the third and fourth decades of life.
4. Higher mortality and seen in elderly patients with perforative pertitonsis.
5. Outcome for colonic perforation is poor.
6. Patients managed conservatively has poor outcome.
7. Mortality is higher in Ileal perforation as compaired to Duodinal perforation.
8. Compared to other perforative peritonitis appedndicular perforation has a better outcome.
9. Complications are higher for ileal perforation, followed by appendicular and duodenal perforation.

10. Morbidity could not be assessed in gastric and colonic perforations due to less number of cases.

11. Ileal perforation patients have the longest hospital stay.

12. Size of perforation does not alter the final outcome of perforative peritonitis.

13. Associated comorbid factors in perforative peritonitis increases mortality.

14. The time between perforation and presentation is inversely propotional to the final outcome.

15. Surgery is the definitive treatment for all perforative peritonitis.

16. Conservative management for perforative peritonitis cannot be recommended, though it has some value in patients, who are not fit for surgery.

INTRODUCTION

HISTORICAL REVIEW

SURGICAL ANATOMY

REVIEW OF LITERATURE

AIMS OF STUDY

MATERIALS AND METHODS

RESULTS

DISCUSSION

CONCLUSION

PROFORMA

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MASTER CHART

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PROFORMA

Study of Non traumatic Gastro Intestinal Perforation

Name : Age : (IPNO)

Address :

Occupation : D.O.A.

D.O.S.

D.O.D. Outcome-Death / Recover

History

Pain :

Fever :

Vomiting :

Bowel Habits :

H/o Peptic Ulcer :

Associated Factors : Smoker / Alcoholism / NSAID

H/o systemic illness

Examination

G.E.

Pulse :

Bp :

Temp :

P/A Tenderness

Rigidity :

Signs of Dehydration :

Liver dullness :

Shifting Dullness :

Bowel sounds :

Investigations:

B. Urea

S. Creatinine :

S.Electorates :

B. Widal

Culture

X-ray abdomen erect including diaphragm :

X-ray Chest PA view :

USG abdomen

Urine output

Duration of perforation on admission

Resuscitation – IVF

Antibiotics

Intervention:

Laparotomy Frank drainage

Findings

Site

Size

Peritoneal exudete – How much

Culture & Sensitivity

Procedure done

P.O. Period

- Pulse Bp Urine Output Temp

IVF

Antibiotics

BI. Transfusion

Morbidity

Mortality

Wound Complication

Respiratory complications

Biliary leak/ Fecal fistula

Infection / abcess Sinus Wound dehiscence

Intra periotoneal Abscess – Subphrinic – Pelvic

Pus Culture and Sensitivity

Condition on discharge

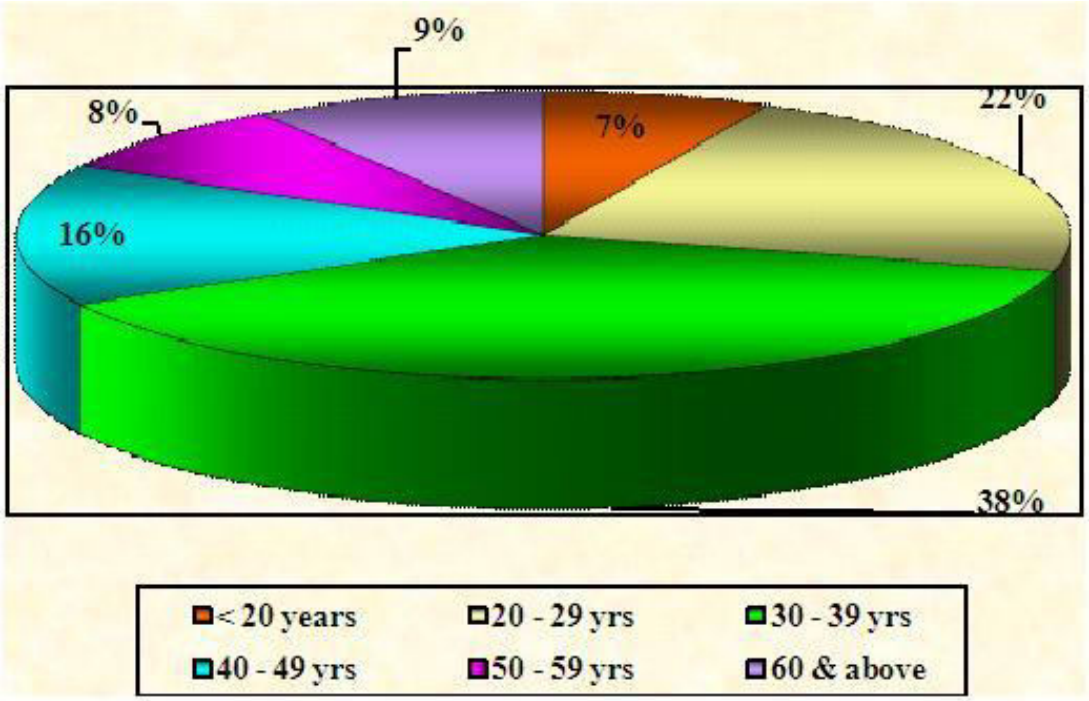
ABBREVIATIONS

M	-	Male
F	-	Female
Du	-	Duodinal Perforation
I	-	Ileal Perforation
A	-	Appendicular Perforation
G	-	Gastric Perforation
C	-	Coloni Perforation
DM	-	Diabetic Melitus
HT	-	Hypertension
S	-	Smoker
A	-	Alcoholic
APD	-	Acid Peptic Disease
LOP	-	Live Omental Patch Closure
PC	-	Primary Closure
JPC	-	Jejunal Patch Closure
R&A	-	Resection and Anastomosis
D	-	Died
R	-	Recovered

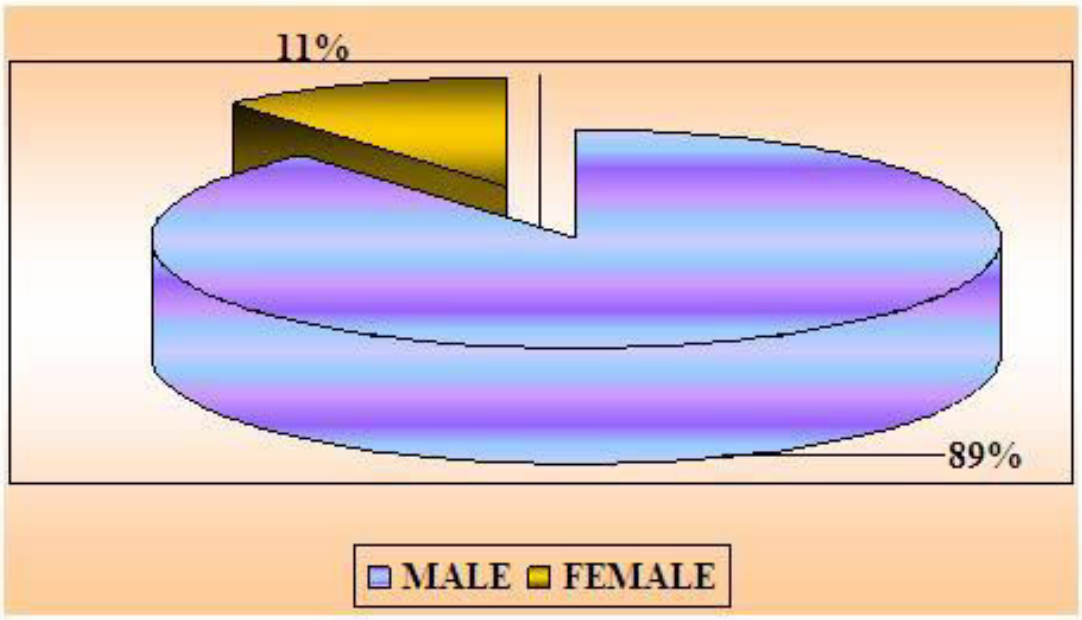
A - Appendicectomy

AD - Abscess drainage

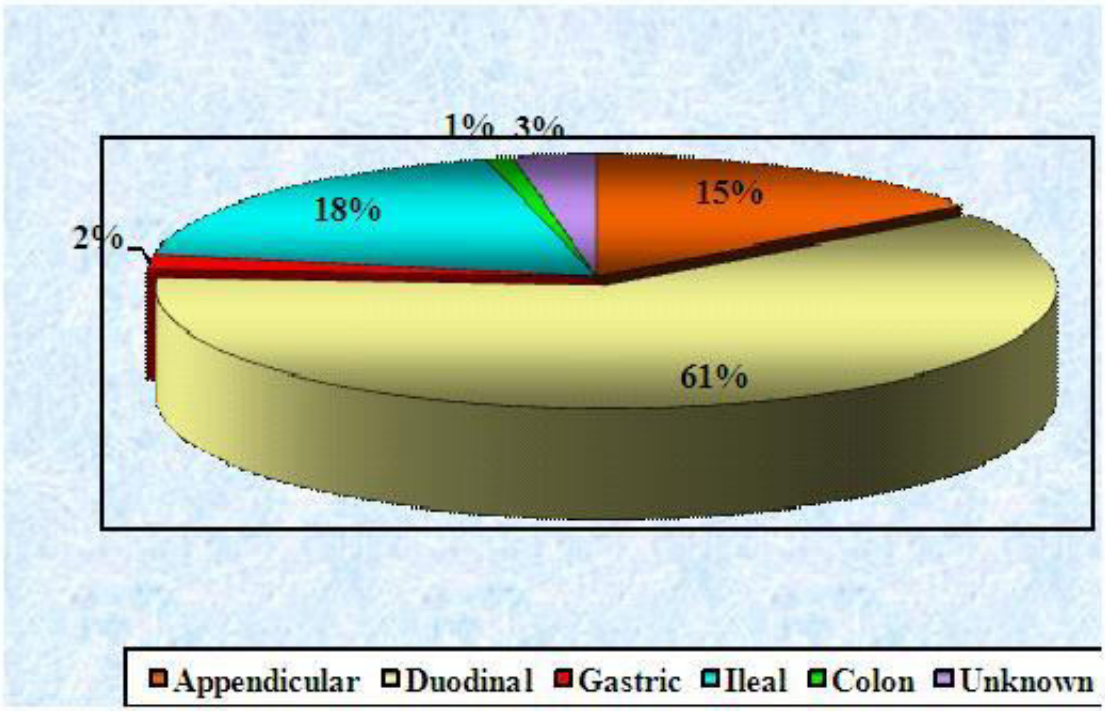
AGE DISTRIBUTION



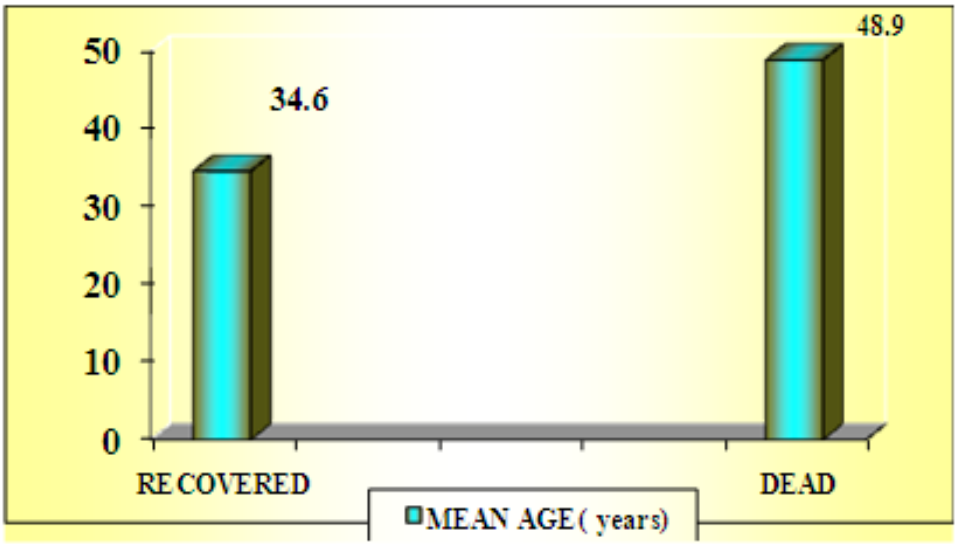
SEX DISTRIBUTION



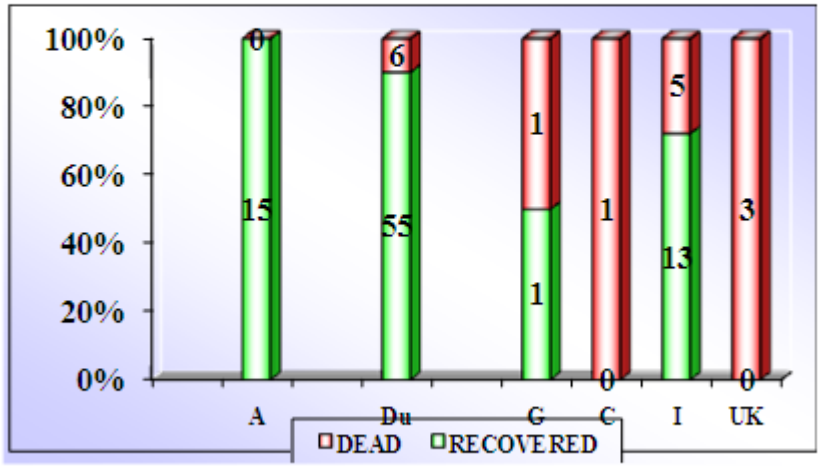
SITE OF PERFORATION



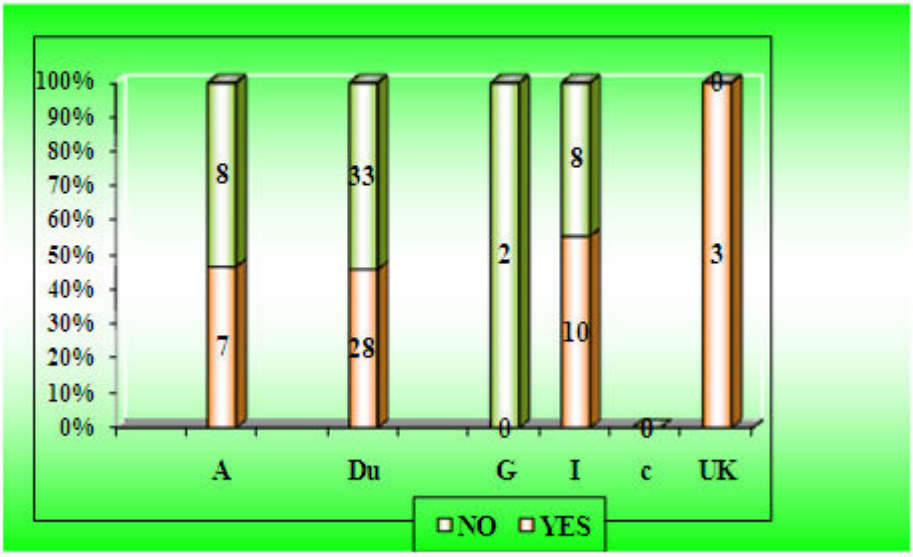
AGE AND OUTCOME



SITE OF PERFORATION & OUTCOME

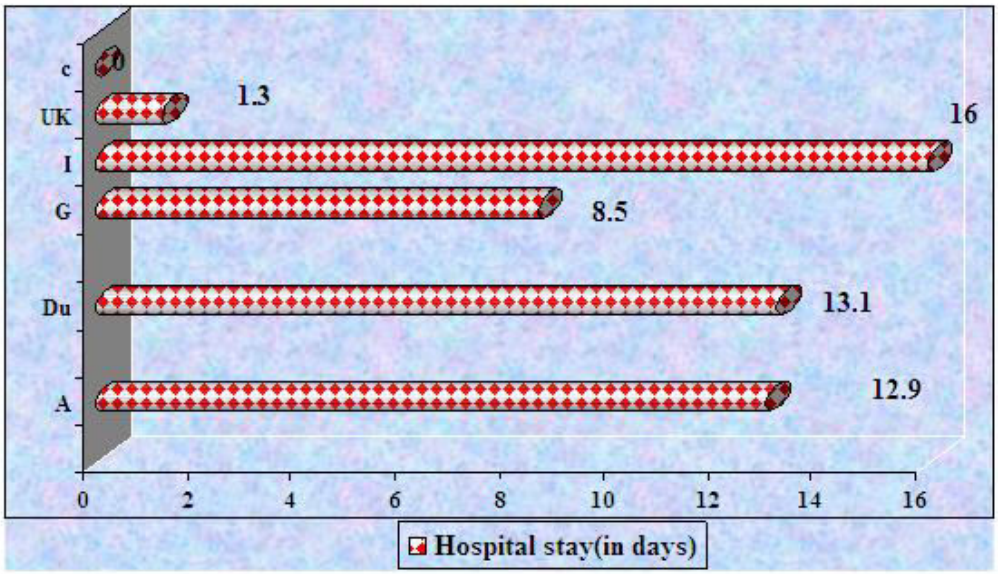


SITE OF PERFORATION & COMPLICATIONS

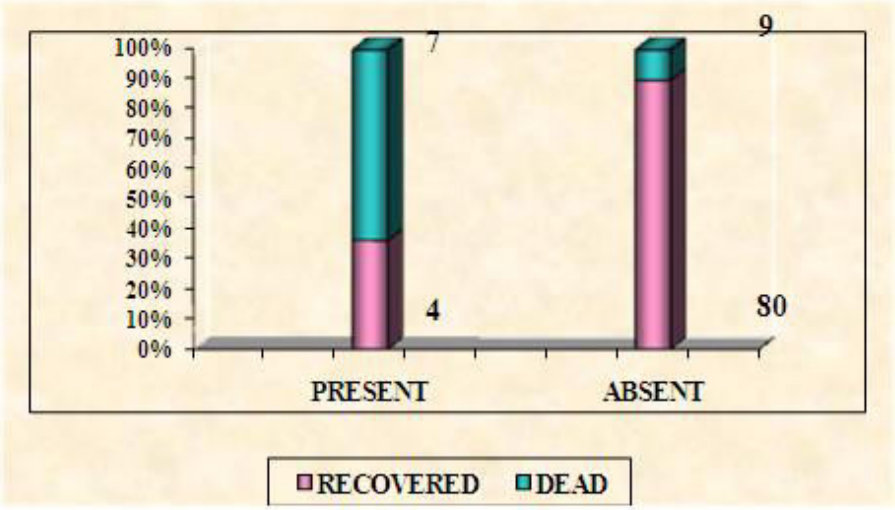


SITE OF PERFORATION AND DURATION OF HOSPITAL STAY

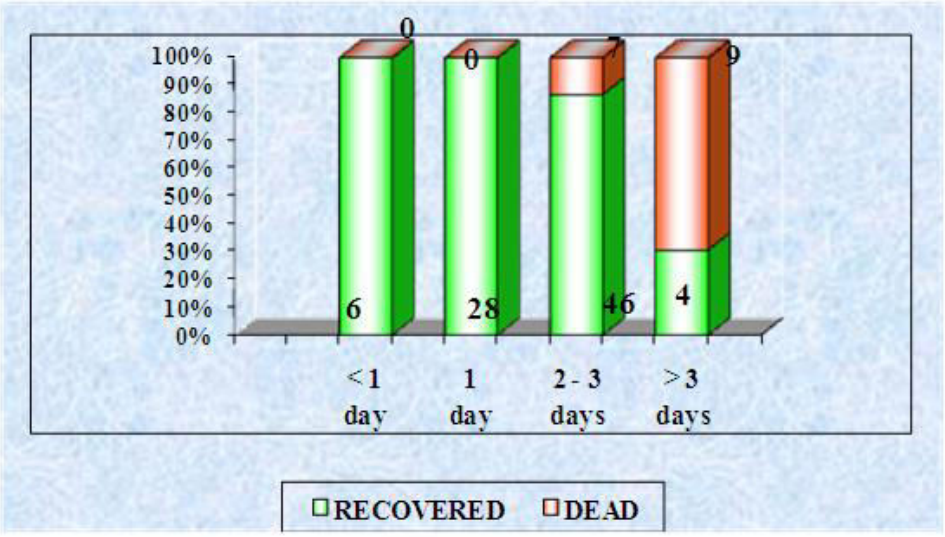
(IN DAYS)



COMORBID CONDITION & OUTCOME



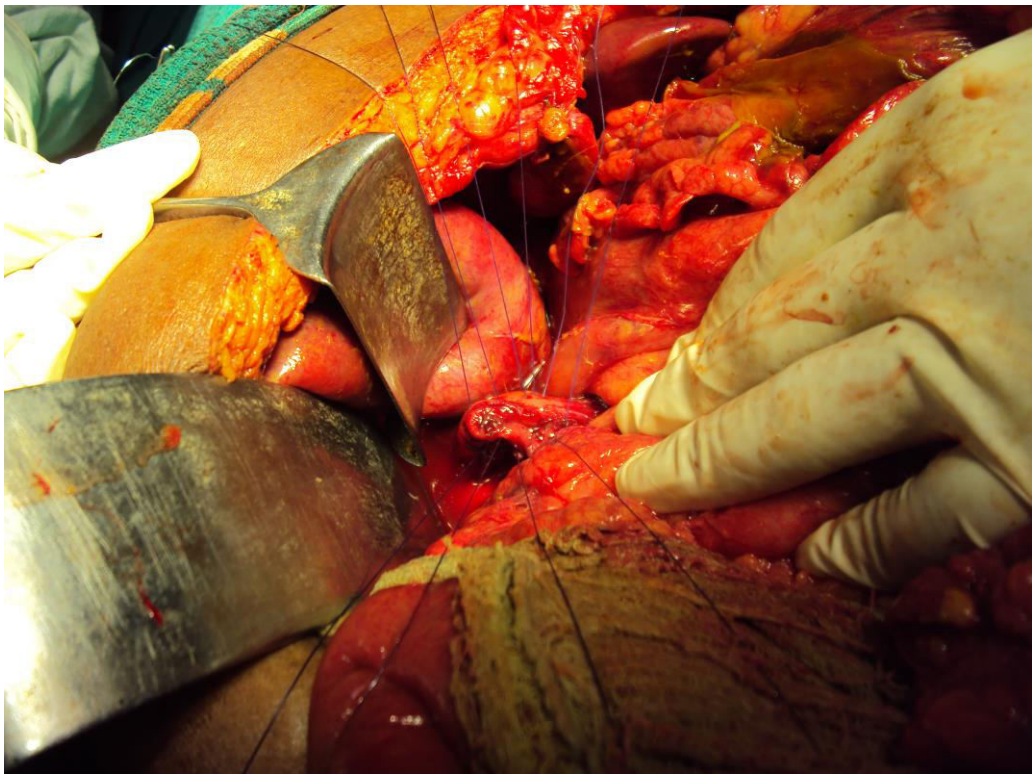
TIME OF PRESENTATION AFTER ONSET & OUTCOME



CXR PA VIEW SHOWING AIR UNDER DIAPHRAGM

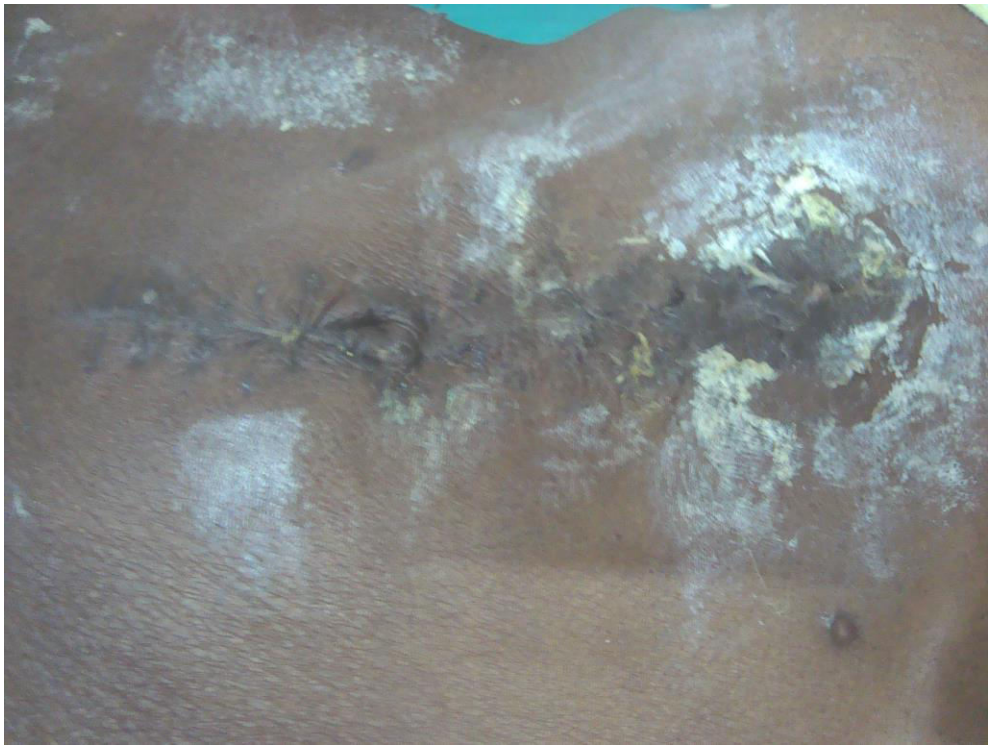
DUODENAL ULCER PERFORATION IN FIRST PART OF DUODENUM

PERFORATION IN FIRST PART OF DUODENUM



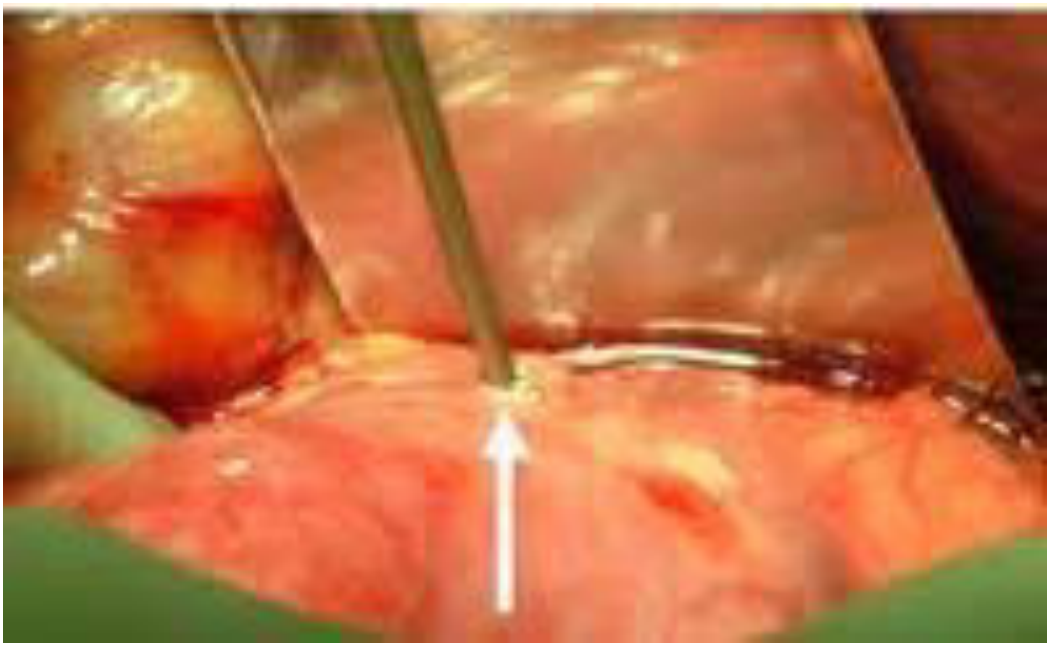
OMENTAL PATCH CLOSURE IS BEEING DONE

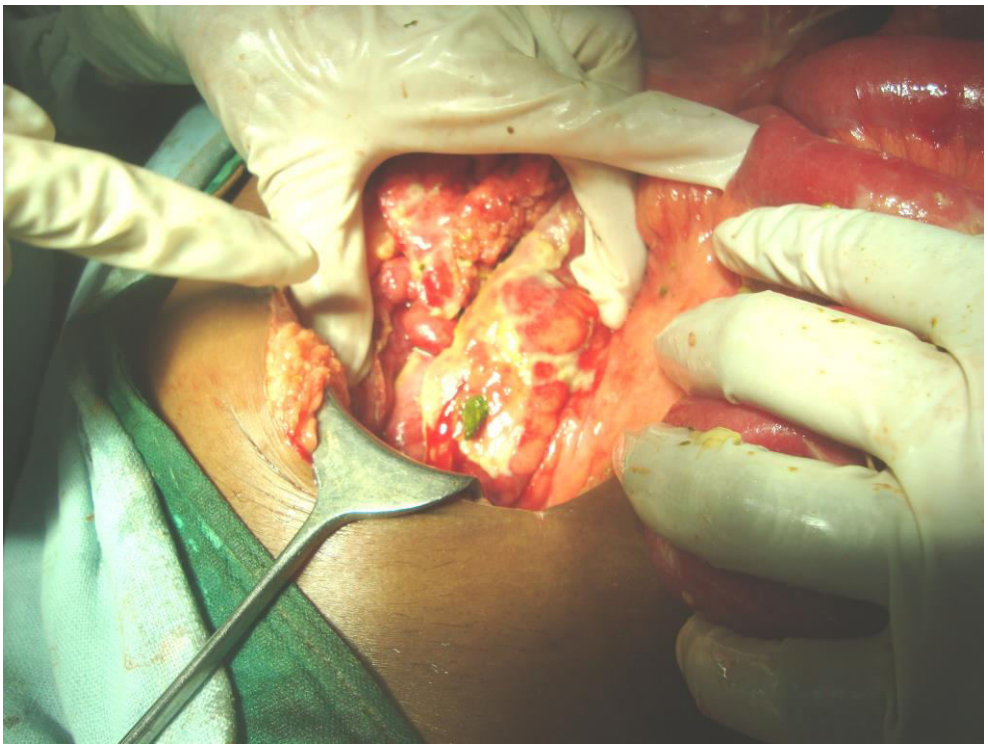
WOUND GAPING FOLLOWING ILEAL PERFORATION



BILIARY LEAK- FOLLOWING A DUODENAL ULCER PERFORATION

DUODENAL ULCER PERFORATION IN FIRST PART OF DUODENUM





ILEAL PERFORATION

ILEAL PERFORATION – CLOSURE DONE

ILEAL PERFORATION